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The Use of 5% Lidocaine for Prolonged Analgesia in Chronic Pain Patients: A New Technique

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Background and Objectives. It has been found that 5% lidocaine with 7.5% dextrose causes irreversible conduction block in animal studies. Our case report subjects allowed us to observe the efficacy of 5% lidocaine for a prolonged analgesia *in vivo*. *Method.* After performing a diagnostic nerve block with 1% lidocaine, 5% lidocaine with 7.5% dextrose was injected into three patients with trigeminal neuralgia, postherpetic occipital neuralgia, and intercostal neuralgia, respectively. The patients were followed for one and a half years. Visual Analog Scale (VAS) scores and the side effects were recorded for each patient. *Results.* One patient received a trigeminal block and one patient received an occipital nerve block. Both patients reported immediate and complete pain relief lasting 14 and 8 months, respectively. One patient, given an intercostal nerve block, received immediate pain relief lasting 5 weeks. None of these patients exhibited any appreciable side effects or complications. *Conclusions.* Our observations suggest that 5% lidocaine may be used safely and effectively for the purpose of prolonged analgesia in selected patients with intractable chronic pain syndromes. *Reg Anesth Pain Med 1998: 23: 96–100.*
Key words: neurolysis, lidocaine, chronic pain.

Chronic pain syndromes can be caused by a chronic pathologic process of somatic or visceral structures or by prolonged dysfunction of the nervous system or have added psychological components (1). The nociceptive origin can often be controlled by oral pharmacologic agents and nerve block therapies. However, nerve blocks with local anesthetics may provide only short-term pain relief for patients, such as those with neuropathic or cancer pain. Subsequently, a neurolytic block of a peripheral or cranial nerve may be considered to alleviate the pain for a longer duration. A neurolytic block involves the use of neurolytic agents to disrupt nerves (2–5). The use of neurolytic agents to control chronic pain has been described for more than 100 years (6). Phenol and ethyl alcohol have been widely used as neurolytic agents; however, their neurolytic effect is variable in efficacy and duration of action. Complications, from the injection of those agents, that may also occur include neuritis, neuroma formation, and the sloughing of subcutaneous tissue, mucosa, or cartilage.

A solution of 5% lidocaine with 7.5% dextrose is an agent typically used for spinal anesthesia (7). Recently, it has been noted that this solution may be associated with a rare postblock effect—cauda equina syndrome (8–10). Some believed that this might be due to the direct exposure of the cauda equina to a high concentration of lidocaine. Lambert et al. used a solution of 5% lidocaine with 7.5% dextrose in desheathed bullfrog sciatic nerves

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and found that it caused irreversible conduction block in these nerves (11). We believed 5% lidocaine could provide longer lasting analgesia when used on peripheral nerves for better long-term control of intractable pain. We reported on our preliminary observations detailing the efficacy of 5% lidocaine as an alternative to neurolytic agents for a longer lasting analgesia in three patients with chronic pain.

Case Reports

Case 1

A 40-year-old white female with progressive multiple sclerosis presented to our pain center with constant sharp, shooting, stabbing, lancinating, and gnawing pain in her right cheek and chin area which began approximately 4 years previous. Her multiple sclerosis began 8 years ago, but the pain started after her wisdom teeth were extracted. Initially, she was placed on hydrocodone, amitriptyline, carbamazepine, and baclofen by a local neurologist, but all of these medications were subsequently discontinued because of adverse effects. She had also tried other types of antidepressants, anticonvulsants, anti-inflammatories, and analgesics as well, but they were ineffective in relieving her pain. She had no other medical problems. Examination revealed severe hyperesthesia and hyperalgesia in the region of the mandibular nerve and slight hyperesthesia and hyperalgesia in the infraorbital area of her right face. Touching her buccal mucosa triggered pain. Speech was laborious because of pain. Motor weakness of the right leg from multiple sclerosis was noted. With the impression of trigeminal neuralgia of the mandibular nerve (V3), she was placed on nortriptyline and choline magnesium trisalicylate. The patient also underwent psychological evaluation and treatment with cognitive-behavioral modalities. We performed a series of mandibular nerve blocks by injecting a mixture of 1% lidocaine and methylprednisolone. The Visual analog scale (VAS) (0 = no pain and 10 = maximum pain) was used to assess the efficacy of analgesia before and after the injection. Pain improved from VAS 10/10 to 3–5/10. However, the pain returned to VAS 10/10 in 3 weeks. The mandibular nerve block with 5% lidocaine was discussed with the patient, and an injection of 1 mL of 5% lidocaine with 7.5% dextrose was given incrementally after a trigeminal paresthesia was elicited by a nerve stimulator. The injection site was observed for 15 minutes to monitor any adverse effects such as swelling, redness, or itching. Immediate and complete pain relief, VAS 0/10, was noted in 1 minute without any significant side effects. Speech also improved with pain relief. The patient experienced slight numbness in the lower lip area 5 minutes after the injection, and it disappeared in 30 minutes. The patient's pain relief was ascertained after 1 day, 1 week, and every 3 months thereafter. There was no appreciable pain, numbness, burning, or motor weakness along with the mandibular distribution. However, the patient began to experience increasing pain along the maxillary division (V2) later. A diagnostic and 5% lidocaine block for infraorbital nerve were performed using the same technique as described earlier. Complete pain relief was obtained 1 minute after a 1 mL of 5% lidocaine with 7.5% dextrose injection. Visual analog scale changed from 8/10 to 0/10 and remained 0/10 thereafter. She was followed after 1 day, 1 week, 1 month, 3 months, and 6 months, and again she remained completely pain-free without any side effects. The patient returned to our pain center 14 months later because pain began to recur to VAS 10/10 in the V2 and V3 distribution, but the pain was more severe in the lateral side of the tongue and lower lip. A repeated block with 1 mL of 5% lidocaine and 7.5% dextrose was performed again to the mandibular nerve and infraorbital nerve. The patient currently reports minimal pain (VAS 0–1/10) and no side effects.

Case 2

A 67-year-old white male patient had chronic left occipitotemporal pain since having a herpes zoster infection two and a half years previous. The patient had been treated with various medications, including amitriptyline, nortriptyline, sinequan, zovirax, carbamazepine, dexamethasone, opioids, and anti-inflammatories, without any considerable benefits for his postherpetic neuralgia. The pain was constant, sharp, shooting, and burning in nature and was associated with allodynia, hyperpathia and/or dyesthesia. His past medical history was not remarkable. Examination showed significant hyperesthesia, hyperalgesia, numbness, and tenderness in the left occipitotemporal scalp area. We discussed further pharmacologic therapy with this patient; however, he declined. After instituting a psychological evaluation and therapy for cognitive-behavioral modification, relaxation training, and stress management, we performed a series of occipital nerve blocks and field blocks with 1% lidocaine and methylprednisolone, which only gave a few hours of pain reduction to a VAS of 3/10 each time.

Cryoanalgesia was discussed with the patient and cryoneurolysis of the occipital nerve was performed using Lloyd cryo-probe (Neurostat-Markll, Westco Medical Corporation, San Diego, CA); however, it only relieved the sharp pain for a few hours with VAS 2/10, and the patient reported the pain returned with a VAS of 10/10. Neurolysis with phenol was discussed and 1 mL of 6% aqueous phenol was injected incrementally using a nerve stimulator. After the phenol injection, the patient received good pain relief in 2 days with VAS 2–3/10 and was very satisfied. There was no additional numbness or skin sloughing. The patient returned to our pain center 3 1/2 months later with a recurrence of pain, VAS 10/10. A repeated block with 1 mL of 5% lidocaine and 7.5% dextrose was discussed and performed after a diagnostic occipital nerve block with 1% lidocaine. Good pain relief resulted and reduced the VAS from 9/10 to VAS 1–2/10 in 1 minute. The patient was followed after 1 day, 1 week, 1 month, 3 months, and 6 months thereafter. There were no side effects reported and satisfactory pain relief with VAS 2/10 lasted for the 8 months of follow-up.

Case 3

A 79-year-old white male patient had chronic intractable pain in his right antero-lateral chest wall for 13 months after a herpes zoster infection. The pain was constant, sharp, stabbing, and burning in nature and was associated with significant allodynia and hyperpathia. The patient tried capsaicin cream, zovirax, and various opioids without benefit, and the use of other medications such as antidepressants, anticonvulsants, and steroids was limited because he developed multiple allergic reactions and side effects. His past medical history included coronary artery disease with previous myocardial infarction, gynecomastia, and prostate cancer. Physical examination revealed hyperesthesia and hyperalgesia in the right-sided dermatomes of T2-T4. After a psychological evaluation and several treatment sessions, we performed a series of thoracic sympathetic blocks, and oral choline magnesium trisalicylate was added. Pain improvement was noted with VAS 2–3/10; however, the duration of pain relief became shorter after each treatment, changing from days to a few hours. Cryoneurolysis of the intercostal nerve was discussed and performed using a Lloyd cryo-probe at the level of T2-T4 under fluoroscopic guidance after a successful diagnostic block with 1% lidocaine. Satisfactory pain relief with a VAS of 1/10 lasted for 5 weeks; however, the original pain level of a VAS of 9/10 returned. We discussed and performed an intercostal nerve block injecting 1 mL of 5% lidocaine and 7.5% dextrose during fluoroscopic guidance. Good pain relief with a VAS of 1–2/10 was noted in 3 minutes without side effects. The patient experienced slight numbness for 15 minutes after the injection, but this resolved completely. The patient was followed after 1 day, 1 week, 1 month, and thereafter. The patient obtained good pain relief with a VAS of 1–3/10 for 5 weeks, and the pain has gradually returned.

Discussion

Neurolytic agents are sometimes advocated for interruption of pain transmission in selected patients with neuropathic or cancer pain. The pain from neuropathic origin or cancer is usually severe and may not respond to conservative treatments. Although the use of a neurolytic procedure for peripheral neuropathic pain is controversial, a chemical neurolytic block may be effective in alleviating pain, especially for elderly patients, patients in poor medical condition, and patients considered poor risks due to prior extensive surgical procedures. These blocks are simple to perform and are thus attractive to many physicians.

A variety of agents have been used to achieve chemical neurolysis, such as distilled water, hypertonic saline, ammonium salt, phenol, and ethyl alcohol (12). Phenol and ethyl alcohol have been most frequently used because of their predictable effects and infrequent side effects.

Phenol was first used for trigeminal neurolysis by Putnam and Hampton (13) in 1936. Injection of more than 5% phenol directly into the tissue causes protein coagulation and necrosis with nonselective blocks of nerve fibers, while injection of a lower concentration of phenol (<5%) may produce only a local anesthetic effect.

Ethyl alcohol was first used to treat trigeminal neuralgia in 1902. Concentrations of 50% and 100% ethyl alcohol can cause extraction of phospholipid cholesterol and cerebroside in the neuron and precipitation of mucoproteins and lipoproteins. This results in a separation of the myelin sheath and swelling of the Schwann cell and axon, and eventually destroys the nerve fibers nonselectively (14).

In addition to their neuronal effects, phenol and ethyl alcohol may cause tissue damage after injection, such as sloughing of subcutaneous tissue, mucosa, and cartilage. Ethyl alcohol may cause severe pain during injection and may produce neuritis with intense pain after injection, whereas phe-

nol typically does not cause significant pain on injection. Phenol causes numbness after injection and may require hours to days to show neurolytic effects.

Neurotoxicity from local anesthetics has been reported, but most often it appears to be caused by local anesthetic additives, such as bisulfite or benzyl alcohol. Local anesthetics per se were not often implicated in directly causing neurotoxicity (15). Others (16,17) indicated that at high enough concentrations, local anesthestics may cause endoneurial edema and Wallerian degeneration with Schwann cell injury and axonal dystrophy. Rarely it is also noted that the direct exposure of the cauda equina to a high concentration of lidocaine may contribute to the development of cauda equina syndrome. Lambert et al. noted that exposure of desheathed frog sciatic nerve fibers to 5% lidocaine with or without 7.5% dextrose resulted in an irreversible total conduction blockade (11). Strichartz et al. also found that 5% lidocaine appears to have neurotoxic potential in mammalian A and C fibers, and C fibers may be more susceptible than A fibers (18). It is unclear how 5% lidocaine caused an irreversible inhibition of neural condition. Lambert et al. concluded that it is not due to a residual local anesthetic effect or to membrane lysis. Other animal data (19) suggest that local anesthetic neurotoxicity might be concentration dependent, and some suggest local anesthetics be administered at the lowest possible effective concentration.

Using this background, we speculated that 5% lidocaine be used *in vivo* for the purpose of prolonged analgesia in chronic or cancer pain patients. The observation that 5% lidocaine did not cause histopathologic changes in neural tissue or surrounding tissue also suggested the drug may offer advantages for peripheral neurolysis. We successfully used 5% lidocaine with 7.5% dextrose, which is currently and commercially available as a spinal anesthetic agent for the purpose of prolonged peripheral nerve analgesia in three patients. Our data showed a variable duration with 5% lidocaine; however, it appears to last weeks to months. Two of our patients who showed trigeminal division paresthesia from a nerve stimulator received immediate, long lasting and complete pain relief, 14 and 8 months, respectively. One patient without paresthesia had a shorter duration of pain relief of 5 weeks. Further studies and follow-up are necessary to determine the typical duration of neurolysis. None of these three patients showed any subcutaneous tissue damage, neuritis, increased paresthesia following injection, and none exhibited pain on injection.

Conclusion

Five percent lidocaine was used successfully to minimize pain in three patients with chronic intractable pain, and it did not appear to cause significant side effects during or after injection. Five percent lidocaine may be considered as an alternative to neurolytic agents for prolonged analgesia. It is less costly to obtain, readily available, and appears to have minimal side effects. Even though only a limited amount of 5% lidocaine can be used for injection, we believe it can be applied to a variety of areas to produce prolonged analgesic effects. Further studies are necessary to assess the appropriate role for 5% lidocaine in managing patients with chronic pain.

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